**Allergic Rhinitis**

**Incidence and Prevalence**

Allergic rhinitis is the 6th most prevalent chronic disease condition in the United States of America. The condition affects approximately twenty percent of the American population, usually beginning before the age of 30 years. The incidence and prevalence continually decreases with time, with the condition having a mean occurrence age of ten years (Chapel *et al.,* 2014). The condition has an equal predominance ratio in males and females alike. The genetic correlation of the condition is complex, but strong genetic relationships and incidence has been noted.

**Pathophysiology**

Allergic rhinitis is an IgE mediated hypersensitivity response mediated by mast cells. Among the allergen triggers of the hypersensitivity reaction are animal and plant proteins including pollen grains, molds, saliva, and urine among others. Thee pathophysiology of the condition is related to risk factors such as family history, repeated exposure, and environmental presence of allergens (Kumar, 2015).

The initial step in the pathogenesis of the condition involves a phase of sensitization in which exposure to an allergen results in activation of Type 2 T Helper cells, and IgE class switching in B cells. Consequently, there is production of IgE that bind to on mast cells (Chapel *et al.,* 2014).

The immediate reaction that characterizes Allergic rhinitis occur in repeated exposure to allergens, in which an antibody-antigen reaction initiates a degranulation of mast cells, resulting in the release of not only preformed mediators such as histamine, enzymes (such as chymase, tryptase and hydrolase), and proteoglycans such as heparin,  but also synthesized mediators such as lipid products and cytokines. The lipid products include leukotrienes C4 and D4, prostaglandins D2, and Platelet Activating Factor. Released cytokines include TNF, IL- 1, and various other chemokines (Chapel *et al.,* 2014).

These inflammatory mediators result in the immediate manifestations of the condition, with different mediators leading to different specific responses (Chapel *et al.,* 2014). Histamine results in increased mucus secretion, damage to the enzymes in tissue, leukotrienes in smooth muscle contractions, prostaglandins in bronchospasm, while PAF leads to increased vascular permeability and vasodilation. Immediate symptomatic manifestations include edema, mucus secretion, and smooth muscle contractions (Kumar, 2015).

Lately, there is a spectrum of eosinophil, basophil, and mononuclear cell infiltration. Additionally, the condition may be classified as seasonal and perennial based on the climatic conditions and the specific responses by an individual. Seasonal allergic rhinitis is most often misdiagnosed, with references made to a permanent cold.

**Physical assessment and examination**

The physical assessment of patients suspected to have with allergic rhinitis involves the identification of characterizing signs and symptoms. The signs and symptoms include nasal stuffiness and congestion, pale and boggy mucous membranes, a continuous sensation of plugging of the ears, sneezing, presence of nasal polyps, watery eyes, and presence of long eye lashes among others (Goubau *et al.,* 2013). The physical assessment would additionally involve eliciting the presence of constitutional symptoms such as fever fatigue dullness, occasional disorientation, and other signs and symptoms associated with sleeping difficulties. Physical examination of the patient would involve the cardiopulmonary system, as the condition would affect the lung and cardiac functions. Visual, auditory, pulmonary, and cardiovascular assessments would be involved in the process.

**Evidence-based treatment plan**

The treatment plan for Allergic rhinitis involves medications, patient education, follow up, and referral if need be.

**Medications**

 this involves the use of antihistamines such as cyclizine at a dosage of 25-50 mg. Also decongestants as pseudoephedrine could be used. Additionally, nasal spray, cromolyn, could be used. In urgent, and various other selective cases, systemic steroids could be used.

**Patient education**

 patient involves advising the patients to limit exposure to the suspected allergens that have previously resulted in a reaction. Therefore, suspected patients are encouraged to keep clean air environments. Patient would also be encouraged to keep cleanliness so as to prevent development of infections (Chapel *et al.,* 2014).

**Follow up**

patients who have experienced a recent reaction would be advised to report back to the health facility every week, for assessment of the progress of the hypersensitivity reaction, and the possibility of development of secondary infections including otitis media (Chapel *et al.,* 2014). In cases where antihistamines are used, patients would be advised to come back to the clinic after two days to monitor the adverse effects including urinary retention, and sedation.

Prophylactic measures are similarly critical in ensuring that patients do not experience acute attacks. Sodium cromoglycate and intranasal corticosteroids could be used (Goubau *et al.,* 2013).

**Referral**

referrals would mostly not be necessary, unless the necessary pharmacological agents are unavailable.

**Any diagnostic studies and laboratory tests**

The presentation of allergic rhinitis is usually similar to other conditions such as nasal polyps and tumors, chronic sinusitis, and the effects of various other pharmacological agents. As such laboratory tests focus on proving sensitization (Chapel *et al.,* 2014). Therefore, skin tests would form part of the primary laboratory test. Also, Radioallergenosorbent tests would confirm cases of suspicion, by helping differentiate allergic rhinitis from non-allergic causes of similar signs and symptoms (Kumar, 2015).

**References**

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Goubau D, Deddouche S, Reis E, et al (2013) Cytosolic sensing of viruses. *Immunity*. *An excellent review on the numerous* *mechanisms used by cells to recognize viral DNA and RNA.* 38:855–69

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